Guest Editorial

TIA—Treacherously Inaccurate Acronym

Things are seldom what they seem,
Skim milk masquerades as cream.
— W.S. Gilbert,
H.M.S. Pinafore

Herbert L. Fred, MD

What would your diagnosis be in someone who had a focal sensory or motor impairment that came on abruptly, reached maximal intensity within 1 to 5 minutes, resolved in 24 hours or less, and left no residua? If your answer is TIA—transient ischemic attack—it could be wrong in up to 30% of such cases.[1] And if the diagnosis is wrong, the patient's management could be wrong, and the outcome could be costly—physically, emotionally, and financially.

Think of it this way. The diagnosis of TIA is difficult at best and is frequently made from the patient's history, rather than from clinical observation.[2] Patients, however, vary widely in their ability to communicate effectively. Physicians, in turn, vary not only in their ability to elicit and interpret the patient's history, but also in their semantic and operational concepts of TIA.[3] Not surprisingly, therefore, interobserver differences in the diagnosis of TIA are common, even among neurologists.[1,3,4] To complicate matters, tests to confirm TIA do not exist.[1,4] The reason should be obvious: these episodes are so brief and unpredictable that demonstrating focal cerebral hypoperfusion during an actual attack is virtually impossible.[5]

Clearly, then, the diagnosis of TIA is always *presumptive*. Failure to appreciate this fact stifles consideration of causative mechanisms other than ischemia and may ex-

TABLE I. Nonvascular Disorders Mimicking Transient Ischemic Attacks*

Metabolic
- Hypoglycemia[6]
- Hypercalcemia[7]
- Hypocalcemia**
- Hyperkalemia[8]
- Acute intermittent porphyria**

Neoplastic
- Hodgkin's disease[9]
- Meningioma[10,11]
- Astrocytoma[10,11]
- Glioblastoma[10,11]
- Kaposi's sarcoma[12]
- Pheochromocytoma[13]

Infectious
- Toxoplasmosis[12]
- Cryptococcal meningoencephalitis[12,14]
- Lyme disease[15]
- Tuberculous granulomas of the central nervous system[16]

Neuropsychiatric
- Hypersensitive carotid sinus reflex[17]
- Meniere's disease[18]
- Hysteria[1,4]
- Multiple sclerosis[19]
- Epilepsy[4,11]
- Hyperventilation[3,20]
- Anxiety[1]

Miscellaneous
- Cervical spondylosis[21]
- Dural sarcoidosis[22]
- Subdural hematoma[23]
- Volume depletion[1]
- Drug reactions[1,24]

*Excluded are vascular-related mimickers of TIAs, e.g., cocaine abuse, migraine, cerebral amyloidosis, polycythemia, and extramedullary hematopoiesis, as well as disorders traditionally linked to TIAs—cardiac dysfunction, embolic disease, and atherosclerosis of the extracranial cerebral vessels.

**Personal observation.

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From: *Department of Internal Medicine, The University of Texas Health Science Center at Houston, Houston, Texas 77030*

Address for reprints: *Herbert L. Fred, MD, 8181 Fannin, #316, Houston, TX 77054*

plain why so many articles on TIA pay little or no attention to differential diagnosis.

Over the years, I have learned that numerous *nonvascular* disorders can mimic TIAs. The more I search for these disorders—at the bedside and in the medical literature—the more of them I find (Table I[6-24]). And the more of them I find, the more misleading I consider the term TIA to be.

I believe that patients would be better off if we stopped using abbreviations and acronyms altogether and started spending time honing our diagnostic and communication skills. Meanwhile, if we insist on acronyms in the setting discussed here, I suggest TNA—transient neurologic abnormality. Others have used TNA to mean transient neurologic attack.[2] Either way, TNA, in contrast to TIA, defines the problem better, carries no misguiding implications, thwarts premature and presumptive conclusions, and prompts a more thoughtful diagnostic approach.

There should be no doubt that TIA is a TIA—treacherously inaccurate acronym.

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